Arachnoid cyst in a patient with chronic kidney disease: Is it always innocent?

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To the editor:

A 51-year-old male was hospitalized due to incidentally detected deterioration in renal function tests. He had hyperlipidemia and hypertension without a history of cranial trauma/seizures. Also, a family history of seizures was absent. Renal ultrasonography revealed bilateral atrophic kidneys with an echogenicity grade 3. In the fundus examination of the patient with congenital left eye exotropia to investigate hypertensive retinopathy, atrophy of the optic disc of the left eye was detected along with fundus findings consistent with bilateral grade 2 hypertensive retinopathy. Further examination was recommended due to suspicion of compression secondary to the intracranial lesion. Meanwhile, the patient developed temporary syncope after hemodialysis but refused treatment and left the hospital. The patient, who presented to the emergency department with uremic findings 2 months later, was taken to emergency hemodialysis upon detection of urea: 200 mg/dl, creatinine 9.5 mg/dl. An arachnoid cyst (AC) was detected in non-contrast cranial CT and cranial magnetic resonance imaging [Figure 1, 2]. Neurosurgically, it was evaluated as congenital type 3 AC, and urgent intervention was not considered. The patient, who continued on the 3/7 hemodialysis program, applied to the emergency department 4 months later with a complaint of right focal tonic contraction that occurred suddenly before the hemodialysis session and lasted longer than 5 minutes. The eyes were open spontaneously, the patient was non-cooperative, the light reflex was +/+, the right eye deviated outwards while the eyes were in a neutral position (sequela sign), and there were tonic contractions in 4 extremities. He was intubated and admitted to intensive care due to status epilepticus. He was followed up with midazolam and levetiracetam treatment and was discharged when his neurological condition improved.

ACs constitute 1% of benign, non-traumatic intracranial lesions [1]. It can be congenital (mostly) or familial [2]. Approximately half of these cysts are located in the middle cranial fossa. In cyst pathophysiogenesis, hypotheses such as abnormalities occurring in the separation or duplication of the arachnoid membrane during embryological life, the osmotic pressure difference between the arachnoid cyst/space, the presence of Aquaporin 1 receptors in the cyst membrane, and the accumulation of cerebrospinal fluid (CSF) between the arachnoid membrane leaves have been put forward. It is usually asymptomatic, but over time, it may become symptomatic with the increase in CSF accumulated in the cyst, resulting in cyst expansion. Neurological symptoms due to arachnoid cysts are observed in only 5% of the patients. In a study in which patients with focal epilepsy were evaluated retrospectively, the presence of AC was found to be higher than in healthy volunteers [3]. However, Brutto et al. could not find a significant relationship between supratentorial ACs and seizures or epilepsy [4]. AC can serve as a basis for very serious clinical conditions such as status epilepticus, as in our patients. The determinant in the AC clinic is usually mass compression, and the increase in intracystic fluid due to cyst membrane fragility and rupture caused by micro or macro cranial traumas may potentiate this effect.

Although they are mostly considered "benign" in general population studies, incidentally detected ACs in CKD cases-especially large ones, should be followed closely, and it should be kept in mind that they may cause serious clinical problems in this population, which is already at high risk for seizures.

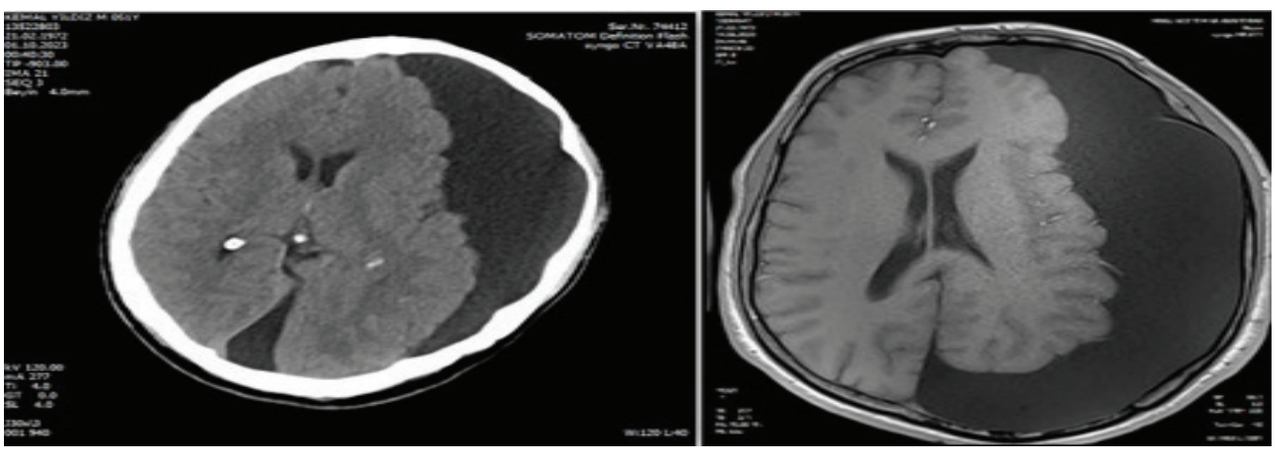

Figure 1. A hypodense area measuring 15x11 cm at its widest point, covering most of the left cranial hemisphere, which may be compatible with an arachnoid cyst pushing the cerebral parenchyma to the right due to CSF density. There is remodeling secondary to cyst compression in brain parenchymal anatomy.

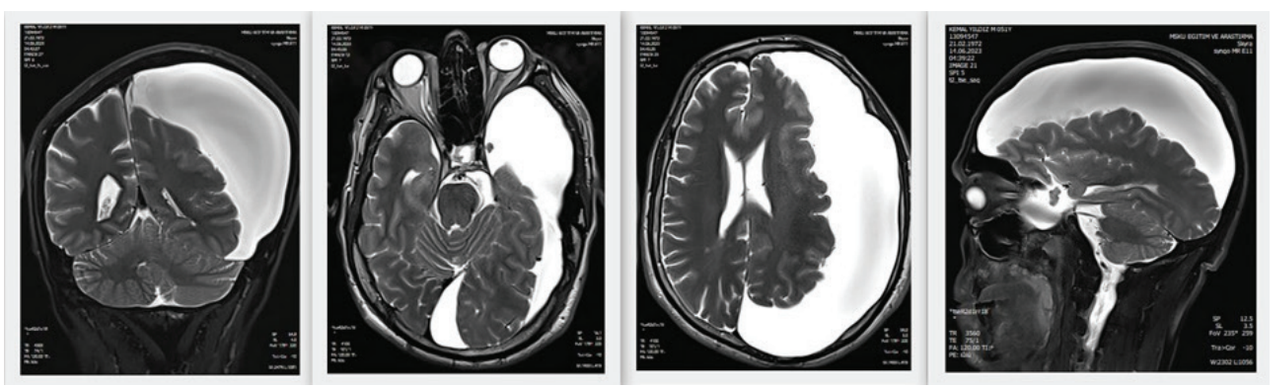

Figure 2. An extraaxial, 6x18x12 cm, asymmetric expansion area was observed in the left cerebral convexity, compressing the brain parenchyma. Appearance is consistent with an arachnoid cyst covering half of the left cranial hemisphere. Cerebral parenchyma is pushed to the right

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